



In vivo porcine left atrial wall stress: Computational model

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ABSTRACT

Most computational models of the heart have so far concentrated on the study of the left ventricle, mainly using simplified geometries. The same approach cannot be adopted to model the left atrium, whose irregular shape does not allow morphological simplifications. In addition, the deformation of the left atrium during the cardiac cycle strongly depends on the interaction with its surrounding structures. We present a procedure to generate a comprehensive computational model of the left atrium, including physiological loads (blood pressure), boundary conditions (pericardium, pulmonary veins and mitral valve annulus movement) and mechanical properties based on planar biaxial experiments. The model was able to accurately reproduce the *in vivo* dynamics of the left atrium during the passive portion of the cardiac cycle. A shift in time between the peak pressure and the maximum displacement of the mitral valve annulus allows the appendage to inflate and bend towards the ventricle before the pulling effect associated with the ventricle contraction takes place. The ventricular systole creates room for further expansion of the appendage, which gets in close contact with the pericardium. The temporal evolution of the volume in the atrial cavity as predicted by the finite element simulation matches the volume changes obtained from CT scans. The stress field computed at each time point shows remarkable spatial heterogeneity. In particular, high stress concentration occurs along the appendage rim and in the region surrounding the pulmonary veins.

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1. Introduction

Progresses in computational methods have given rise to models that simulate the complex three-dimensional, multi-physics electromechanical behavior of the heart and provide detailed spatial and temporal data that cannot be easily obtained experimentally (Bassingthwaight et al., 2009; Hunter et al., 2003). Up to now, the majority of the computational models of the heart have concentrated on the study of the left ventricle, often using standardized three-dimensional geometries (Costa et al., 1996; Taber, 1991; Hunter et al., 2003; McCulloch et al., 1992; Guccione et al., 1995). This approach is justified by the geometry of the ventricles and the small inter-patient geometrical variability; however, the same assumptions are not valid for the atria. The left atrium (LA) geometry cannot be easily assimilated to a regular shape as can be seen in Fig. 1. In panel (a) the three-dimensional geometry of the atrium is depicted at two instants in the cardiac cycle, corresponding to its maximum and minimum volume. The left atrial appendage (LAA, green in Fig. 1) has an irregular shape that varies greatly throughout the cardiac cycle

and does not seem to follow a simple volumetric expansion law. The displacement of the atrium, in general, is non-homogeneous and does not lend itself to convenient geometry simplifications. Moreover, the geometry of the left atrium may change significantly due to pathology, such as sustained elevated pressure in the heart chambers and atrial fibrillation (Heist et al., 2006).

The atrium control ventricular filling through three functional stages: (a) reservoir phase, when the atrio-ventricular valve is closed and pressure builds in the atrium in concomitance with volume growth in the cavity, (b) conduit phase, after the opening of the valve, which allows the blood collected in the atrium to flow freely towards the ventricle, together with the constant stream of blood coming from the pulmonary veins, and (c) active phase, during late ventricular diastole, when the atrium wall actively squeezes the blood still trapped in the atrium towards the ventricle, just before the atrio-ventricular valve closes. Approximately 85% of the atrial cycle is passive; the active part of the cycle pertains to the final 15% of the cycle (phase c) (Hitch and Nolan, 1981).

The time-dependent pressure load due to the blood inside the heart determines a state of mechanical stress in the cardiac wall, the level of which varies locally and across the thickness and is dependent on many factors, including the mechanical properties of the wall tissue and the instantaneous tension acting locally.

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